A rare case of tamoxifen induced bilateral optic neuritis

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SUMMARY

Tamoxifen, an oral medication that blocks estrogen activity, is frequently prescribed for the treatment of advanced breast cancer and as an additional therapy following surgical removal of early stage disease. A 45-year-old female with a history of breast carcinoma treated with tamoxifen presented with sudden onset bilateral visual impairment for 4 days. On ocular examination, the patient exhibited optic disc edema with hyperemia and bilateral anterior pathway defects in visual evoked potentials. Magnetic resonance imaging revealed a thickened right optic nerve sheath with patchy enhancement of the left optic nerve sheath. The patient was diagnosed with bilateral optic neuritis and treated with intravenous methylprednisolone, which resulted in significant improvement in visual acuity and resolution of optic disc edema. This case underscores the importance of vigilant ophthalmological monitoring in patients undergoing tamoxifen therapy to facilitate the early detection and management of ocular complications.

KEYWORDS:
Tamoxifen, Optic disc oedema, Optic neuritis

INTRODUCTION

Tamoxifen, an oral anti-estrogen, is commonly prescribed for the treatment of advanced breast cancer and as adjuvant treatment following surgical resection of early-stage disease.[1] The link between tamoxifen and eye issues was initially noted in 1978. The incidence of tamoxifen-related eye complications varies between 6.3 to 12%. Originally observed in female breast cancer patients receiving exceptionally large doses of tamoxifen (120–130 mg/day), subsequent findings have revealed that ocular problems might also arise with conventional low-dose tamoxifen treatment.[2]

Ophthalmic manifestations associated with tamoxifen include intraretinal crystalline deposits, frequently accompanied by macular edema, keratopathy, pseudocystic foveal cavitation cataracts, and optic neuritis.[3] Optic neuritis constitutes an uncommon but potentially irreversible visual impairment caused by tamoxifen-induced ocular toxicity. It might affect both eyes simultaneously and may manifest as soon as three weeks after initiating tamoxifen therapy.[2]

Although the precise frequency and seriousness of tamoxifen-induced eye conditions remain uncertain, the extensive administration of the medication among patients with breast cancer underscores the actual necessity for recognizing potential adverse ocular outcomes.

CASE PRESENTATION

A 45-year-old woman experienced an abrupt onset of reduced vision in her right eye, which was subsequently followed by diminished vision in her left eye over the past four days. She had a past history of breast carcinoma diagnosed three years back for which she was operated and receiving Tamoxifen 40 mg/day as an adjuvant therapy. The patient had no history of fever, altered bowel habits, smoking, alcohol consumption, intake of other systemic medications, or trauma.

On ocular examination, visual acuity was counting fingers close to the face in both eyes, and the color vision observed was 0/25. Anterior segment evaluation revealed a sluggishly reactive pupil in both the eyes. Fundoscopy revealed optic disc edema with hyperemia, blurred disc margins, disc elevation, and superior venous pulsation, which were absent in both the eyes. Blood tests, including complete blood count, erythrocyte sedimentation rate, C-reactive protein level, and a comprehensive metabolic panel, were within normal limits. Tests for infectious causes and autoimmune conditions, including antinuclear antibodies and antineutrophil cytoplasmic antibodies, were negative. The Humphrey visual field (HVF) showed constricted fields centrally and dense nasal peripheral field defects in both eyes. Visual evoked potentials revealed bilateral anterior pathway defects. Magnetic resonance imaging (MRI) of the brain showed bilateral and extensive optic neuritis with T2 hyperintensity and perineural enhancement. A neurological opinion was obtained and a diagnosis of bilateral optic neuritis associated with tamoxifen therapy was established. After stopping tamoxifen, the patient was immediately administered intravenous methylprednisolone (1 g) in 500 ml of saline once daily for 5 days, followed by oral prednisolone 1 mg/kg body weight for 11 days, 20mg for 1 day, and 10 mg for 1 day and stopped. Symptomatic improvement was noted and after three months, visual acuity improved to 6/6 in both eyes with resolution of the optic disc edema.

DISCUSSION

Tamoxifen belongs to the class of triphenylethylene nonsteroidal estrogen antagonists. It is used in the treatment of estrogen-dependent disseminated breast carcinomas and as adjuvant therapy post-surgery. The mechanism involves disrupting the binding of estradiol to target tissues by reducing cytoplasmic receptors and competitively inhibiting the receptor site. The pathological mechanism underlying tamoxifen-induced ocular toxicity suggests that tamoxifen might trigger the accumulation of drug polar lipid complexes within the lysosomes.

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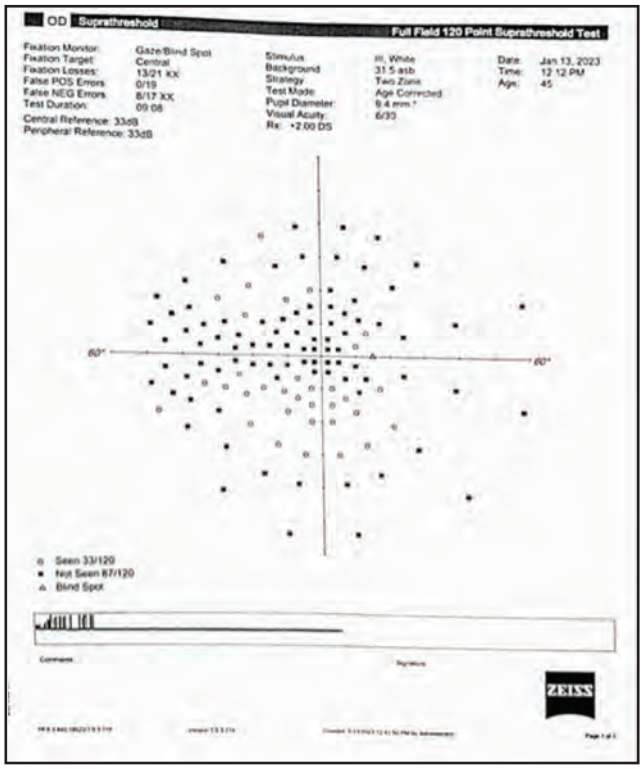

Fig. 1: A) HVF of right eye

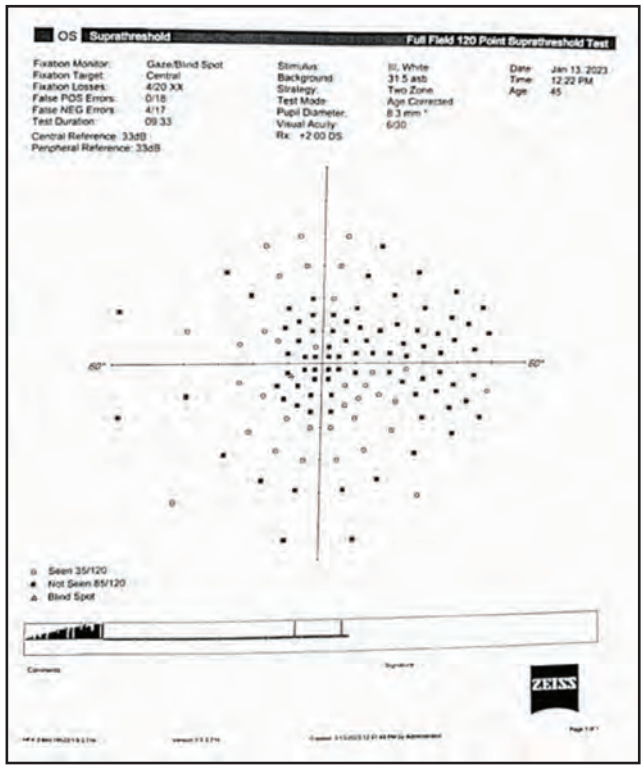

Fig. 1: B) HVF of left eye

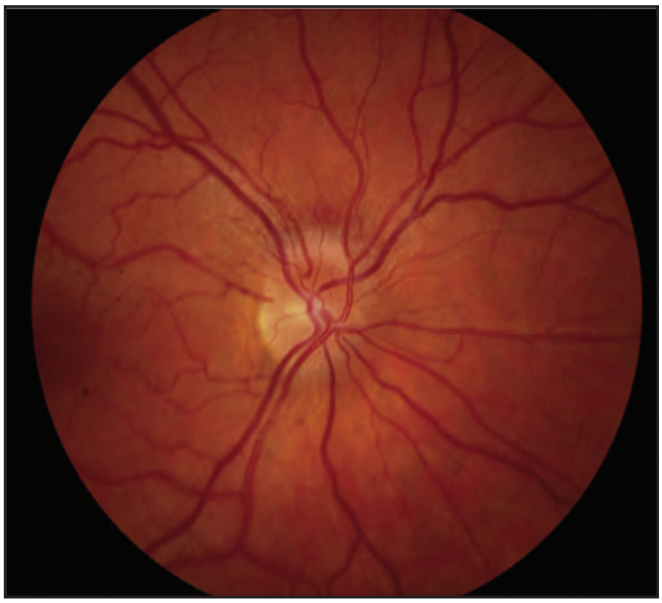

Fig. 1: C) Right eye fundus picture showing disc oedema

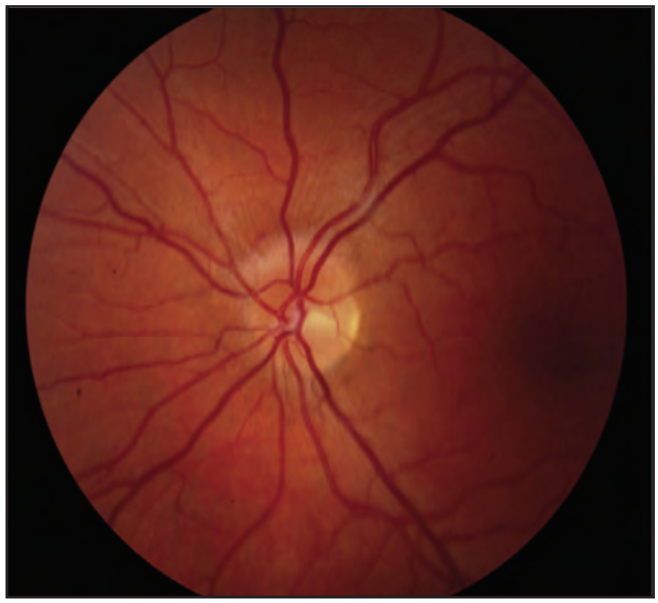

Fig. 1: D) Left eye fundus picture showing disc oedema

The ocular effects of tamoxifen toxicity include whorl-like superficial corneal opacities, retractile crystals in the inner layer of the retina, macular edema, abnormalities in the retinal pigment epithelium, pseudocystic foveal cavitation, and optic neuritis.[3,4] In most documented cases, tamoxifen-induced ocular toxicity appears to be reversible. Cessation of the medication led to enhanced visual acuity and resolution of macular edema, retinal hemorrhage, and corneal alterations.

Several case reports have underscored the ocular toxicity in patients receiving low-dose tamoxifen (10–20 mg BD). Even at these lower doses, tamoxifen therapy can trigger retinopathy, marked by intraretinal refractile crystals and macular edema.[5] The key differentiator between high- and low-dose toxicity lies in the potential reversibility upon cessation of treatment. Several patients prescribed 20 mg of BD daily exhibited regression of retinopathy alongside visual improvement.

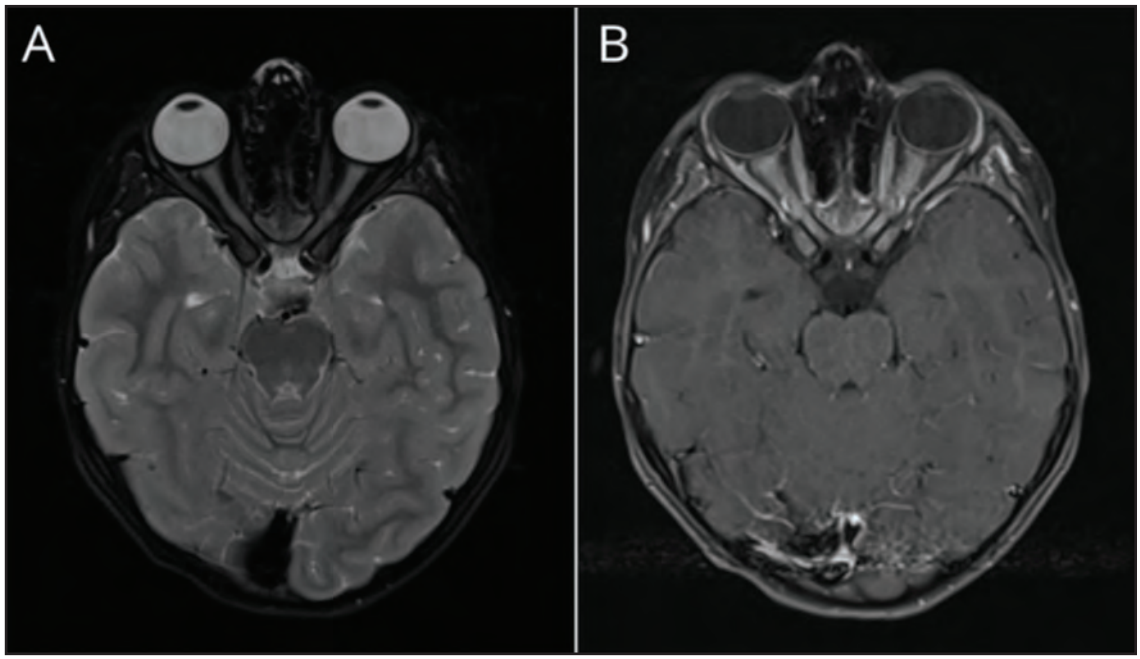

Fig. 2: A) Right eye fundus picture showing disc oedema
B) Brain MRI contrast shows perineural enhancement

Research has indicated that patients undergoing tamoxifen treatment exhibit a decrease in cup volume over time. It has been hypothesized that estrogen plays a neuroprotective role. As a result, the utilization of tamoxifen may potentially disrupt astrocytes located within the glial cells of the optic cup, where estrogen receptors are situated at the optic nerve head. This disruption may induce swelling of the optic nerve head, ultimately resulting in a reduction in cup volume.[2]

In this case, both eyes had disc elevation of one disc dioptre with blurring of the nasal, superior, and inferior disc margins in the right eye and blurring of the nasal and superior disc margins in the left eye. The patient's visual acuity improved considerably within a week of starting corticosteroids, and further improvement was noted over the following months. However, some residual peripheral visual field defects remained, which emphasizes the potential for lasting visual impairment, even with timely treatment. Regular follow-up is crucial for monitoring recurrence and managing long-term sequelae.

However, it is vital to exclude other potential causes that can produce an optic disc appearance similar to optic neuritis, such as carcinomatous meningitis, pseudotumor cerebri, intracranial metastasis, and superior sagittal sinus thrombosis due to metastasis and hydrocephalus. Therefore, thorough neurological examination, lumbar puncture, and computerized tomography (CT) scans are essential for proper evaluation.

CONCLUSION

This case highlights the need for careful ophthalmological monitoring in patients with breast cancer undergoing tamoxifen therapy, as tamoxifen could be responsible for visual impairment in these individuals. Prior to commencing tamoxifen therapy, patients should undergo comprehensive ophthalmic assessment and should be provided with explicit instructions to promptly report any minor visual symptoms. Therefore, prompt recognition and management of ocular complications, such as optic neuritis, can lead to favorable visual outcomes and improve the overall quality of patient care.

DECLARATION

The authors declare no conflict of interest.

INFORMED CONSENT

Written informed consent was obtained from the patient for publication of this case report.

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